Surgical Treatment of Brown Spider Bites

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THE BITE of the brown spider, *Loxosceles reclusa,* may cause localized gangrene, systemic toxicity and even death. Hundreds of bites are treated in physicians' offices and do not require surgical care, but gangrenous lesions, even small ones, may take many weeks and sometimes months to heal. Although gangrenous lesions are typical and the patient saw the spider and was aware of the bite, doctors have not realized their significance and potential hazards.

This article reviews pathogenesis and methods of treatment. Success with early surgical excision and prompt closure of the wound (Table 2) led to a review of 17 hospital cases. Twelve patients required operations.

Historical Review

Spider bites resulting in fever, hematuria and jaundice were reported from Tennessee in 1872,[2] and Kansas in 1929.[11] Gangrene of the skin resulting from spider bites was first recognized in Chile in 1937[8] and the insect was identified as *Loxosceles laeta,* a brown spider related to the *Loxosceles reclusa* found throughout the central and southern United States. The danger of brown spider bite was unrecognized until Atkins, Wingo and associates, in a classic clinical and experimental paper[1] identified this spider as the cause of gangrenous lesions. Hemolytic anemia resulting from *L. reclusa* bites was reported in 1961[9] and deaths have been reported.[6, 10]

The brown spider, *Loxosceles reclusa,* is widely distributed through the central and southern United States, as illustrated in Figure 1. Cases have been reported from all of these states except Alabama and Louisiana. There are some indications that the range of the spider is spreading. *L. reclusa* and a case of necrotic arachnidism was recently reported from California.[7]

L. reclusa is easily identified by the dark brown violin-shaped mark (Fig. 2) extending from the eyes posteriorly on the cephalothorax. This mark is found on no other species. The spider builds small irregular webs out-of-doors throughout the southern states. In cooler parts of its range, such as Missouri and Illinois, it frequently lives indoors. Two of our patients were bitten in bed, and four when donning work clothes in tool sheds, garages, etc. The spider is small, shy and does not attack unless threatened. Captive spiders bite the skin of animals only when teased while they cannot escape. The severity of the lesions resulting from the bite is unpredictable. Necrosis may be minimal or extensive. Twelve of our cases developed gangrenous lesions, large enough to require hospitalization and operation. In animal inoculations, the severity of the gangrenous lesion is proportional to the dose of venom inoculated.

Pathogenesis and Pathology

Experimental animals, including dogs, rabbits, guinea pigs, have been used for quantitative and systematic study not possible with human patients.[1, 3, 5]

The venom is necrotizing, hemolytic and contains a spreading factor, possibly hyaluronidase.

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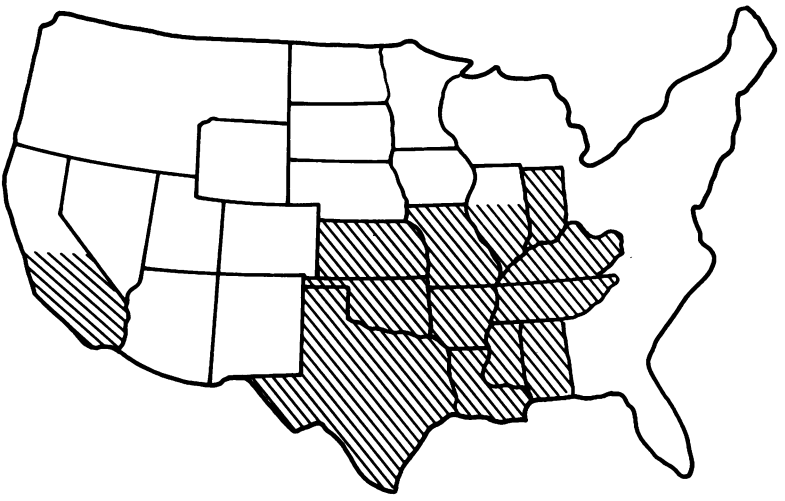

FIG. 1. The geographical distribution of the brown spider, *L. reclusa.*

Severe systemic symptoms, nausea, vomiting, chills, severe malaise, aches and pain may develop in 12 to 24 hours in humans. The mechanism of this is not clear. The systemic symptoms vary according to dose and route of inoculation but are not always proportional to the severity of the skin lesions. Hemolysis, hemoglobinuria, renal failure and hemolytic anemia may develop in 12 to 24 hours after severe bites.[4, 5] At Barnes Hospital in 1954, a young farmer died with hemolysis and renal failure and a gangrenous area of skin erroneously attributed to the black widow spider. One patient in the present series had severe hemolysis, but no renal complications (Table 1, Case 6). These complications can be reproduced experimentally by intravenous inoculation of small doses of the venom in dogs.[3] The most severe reactions and most fatalities occur in children. Blood platelets may be low, and some petechiae and abnormal bleeding may appear. Petechiae may appear in 12 to 24 hours, but are seldom observed in human patients except in the most severe instances. Petechiae appeared distal to a venous tourniquet in two of our patients. A generalized rash may appear in 12 to 24 hours and last for several days. It is macular and erythematous. The rash was seen in four of our patients, including several who healed without necrosis.

The necrotizing component of the toxin causes a gangrenous skin lesion which is pathognomonic (Figs. 3, 4, 5). Inoculation of moderate doses of venom in the skin of rabbits and guinea pigs [1, 5] results in hemorrhage in the dermis, capillary stasis and thrombi within a few hours. Twenty-four hours later there is necrosis of epidermis and dermis at the center and a spreading

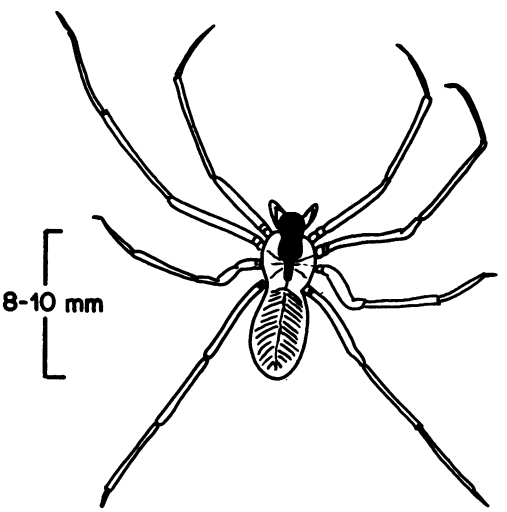

FIG. 2. Diagram of *Loxosceles reclusa.* The dark brown violin-shaped mark on the cephalothorax is found in this species only. The body is 8 to 10 mm. long.

TABLE 1. *Hospitalized: No Operation Needed*

Case and Age	Spider	Bite	Rx	Systemic Symptoms	Rash	Early Lesion	Time Until Healed
1. M. L. 64	"Brown"	Rt. flank	ACTH gel i.m. steroids p.o. <6 hr.	"Tight chest" dyspnea	None	Slight pain and sting, erythema 7 cm. diameter, edema 2.5 cm. diameter	Hosp. 3 d., no necrosis
2. S. S. 50	*L. reclusa*	Shoulder (in bed)	Steroids i.m. and p.o. >12 hr.	Fever 102.4, nausea, malaise	Gen'l.	Cyanotic 1–2 mm. diameter, edema 2–3 cm. diameter	Hosp. 1 d., no necrosis
3. G. W. 26	*L. reclusa*	Hand	ACTH i.m. steroids i.m. and p.o.	Malaise	None	Sl. pain, sl. red 1 d.	Hosp. 3 d., no necrosis
4. J. W. 52	Spider	On lateral thigh (when put on overalls in garage)	Local Rx antibiotics OV × 9	Fever	None	Cyanotic blistered skin 2 cm. in diameter later, ulcer 4.5 cm. large nodes in groin	3 months to heal
5. C. W. 41	"Brown"	Medial thigh (when put on pants from clothesline)	>2 d.	None	None	3 × 4 cm. eschar, inguinal adenopathy, large ulcer later,	Hosp. 6 d., sev. mos. to heal
6. J. J. 2	"Brown"	Back (in bed)	>14 hr.	Convulsion, high fever, hematuria, hemoglobinuria, 1 bloody stool	Gen'l.	Red, 10 cm. diameter, ecchymosis 1 cm., edema entire back, slough 4 cm., later, eschar and ulcer 4 cm. diameter	Hosp. 9 d., sev. mos. to heal

zone of hemorrhage, stasis, sludging and thrombi in capillaries and small blood vessels with infiltrations of leucocytes at the advancing edge of the necrotic lesion. The necrosis is deep, undermining and progressive.

Three days after inoculation a central black gangrenous dry slough, with deep coagulation necrosis about it, and focal abscesses at the edge and depth of the spreading zone of injury appear (Fig. 3). In animals, necrosis frequently involves underlying fascia and muscle. Bites in humans differ mainly in that there is deep and wide

TABLE 2. *Early Excision: Second*

Case and Age	Spider	Site	Dates		Time		Lesion at Operation
			Bitten	Hospitalized	Excised Days After Bite	Healed Weeks Post-op.	
7. B. B. 56	Brown (found in privy)	Upper inner thigh	8/20	8/27 to 9/25	8	2	Eschar Stinking gangrene, deep and wide. Fig. 4 Regional adenopathy
8. L. D. 68	Not proven	Upper inner arm	7/9	7/13 to 7/20	5	2	Central hemorrhage and cyanosis Erythema elbow to axilla, subsided after excision
9. R. H. 15	Brown spiders found in basement	Lateral leg (trousers had been in basement)	8/4	8/5 to 8/24	3	3½	Puncture wound of skin Central necrosis of fat Markedly red skin, 6 cm. dia. Widespread faint erythema, subsided after excision

necrosis of subcutaneous fat. Deep fascia (Case 9) or tendons (Case 15) or deeper structures are seldom damaged in humans. Necrosis of fat, however, is deep and wide (Figs. 3, 5) and the edges of the skin are undermined for several centimeters beyond the eschar. Secondary infection appears in the gangrenous tissue in a few days.

Clinical Manifestations

The diagnosis is made early if the spider was recognized. Brown spiders were identified in 12 of our cases. One patient had a typical necrotic skin lesion which was erroneously attributed to the black widow spider. Four cases were included because of typical gangrenous skin lesions even though the bite or spider was not recognized by the patient. Other diagnoses considered at the onset have been cellulitis, bee or wasp sting, and necrotizing anerobic fascitis. Necrotic bites on fingers have been attributed to arterial occlusions.

Initial symptoms may be mild, only a slight sting or itching sensation, with transient erythema at the puncture site. After a latent period of a few hours, a painful red area appears with a pale, mottled, cyanotic center which may blister. The generalized morbilliform macular red rash appears in severe cases. The rash and systemic symptoms are not necessarily followed by a necrotic skin lesion in all patients.

Forty-eight Hours: The earliest evidence of impending necrosis appears in an area of hemorrhage and discoloration which has become confluent and which no longer blanches on pressure or refills after blanching. The pain is very severe, erythema and edema advance far beyond the central area of hemorrhage, mottled cyanosis and pallor. The erythema may be extensive and alarming, extending across the entire back, buttock or thigh. This early erythema subsides the day following excision of the necrotic tissue (Cases 7, 9). Antibiotic drugs are also administered. The lesion spreads by continuity influenced by position, site, motion and possibly by gravity. The eschar which develops later is asymmetrical (Figs. 2, 3, 4). Some patients have multiple bites of varying severity separated by a bridge of normal skin (Cases 11, 14 and 16).

Stage Closure By Suture or Graft.

Early Excision	Specimen	Comment and Result
Excised 7 d. after bite, secondary suture 13 d.	14 × 18 cm. skin and fat, full thickness 1–3 cm. margin of normal fat Fig. 4	Mild secondary infection and lymph drainage
Excised 5 d. after bite, secondary suture 10 d.	5 × 6 cm. piece of skin, full thickness down to deep fascia, including 1–3 cm margin of normal fat with slight edema	Bite not recognized by patient but had typical rash and small lesion. Healed by primary intention; stitches removed in office
Excised 3 d. after bite, split thickness graft 7 d.	Full thickness skin + deep fascia 1–3 cm margin of edematous fat	Earliest excision reported; relieved pain, fever in 24 h. Brown spiders found in basement had bitten another person also

TABLE 3. *Late Excision and*

Case and Age	Spider	Site	Dates		Time	
			Bitten	Hospitalized	Operated Wks. After Bite	Healed Wks. Postop.
10. J. H. A. 10	Brown (in bed)	Lower abdominal wall	6/9 readmit	6/10 to 6/17 7/15 to 7/17	5	1
11. M. M. D. 53	Brown (in bed)	Left inguinal area right thigh	5/12	5/14 to 5/27	3	9
12. S. K. 26	Typical skin lesion	Right ankle (two bites)	8/5	9/10 to 9/21	5	2
13. E. S. 40	Brown	Anterior mid-thigh (inside trousers)	6/26 readmit	8/1 to 8/8 8/27 to 9/6	9	1½

Secondary infection of the gangrenous skin is inevitable and because antibiotic agents are given in large doses, the organisms cultured are various resistant strains of *B. proteus, B. subtilis,* pyocyaneous, aerogenes, and so forth. Infection continues as long as necrotic tissue is present. Death from invasive infection has been prevented by antibiotic drugs but regional lymphadenopathy is frequent and local pain and tenderness persist around the lesion until debridement. Shock and severe septicemia have developed from minor lesions treated for several months in outpatients.[12] A distinctive feature of the necrotic lesions of the thigh, buttock and abdominal wall is the deep and wide necrosis of fat undermining the skin 1 to 3 cm. around the black eschar (Figs. 3, 5). This plaque of indurated fat is readily palpable, the edge is very tender, and severe pain may persist for weeks or until the lesion is excised.

With non-operative treatment, the stages of healing are prolonged for several months. After the black dry skin eschar is formed, the adjacent edge of skin is slightly red, edematous and gradually the viable skin separates from the leathery black eschar (Fig. 3). When this loosens and is lifted off or trimmed away, the infected necrotic fat remains and slowly separates. The ulcer has undermined edges until the process of contracture of granulation and scar tissue diminish the size and depth of the ulcer. The final scar is broad, slightly depressed and irregular or stellate in outline.

Treatment

The best treatment is administration of corticosteroids in the first 6 to 12 hours, later antibiotic drugs and operation when needed. Corticosteroids should be given promptly in large doses because they may be of value in aborting or treating early systemic symptoms. Antibiotics prevent invasive infection and are essential. Antihistaminic drugs and Regitine have also been used for the early treatment because of certain assumptions about the toxin and pathogenesis of the disease. None of these drugs, however, can prevent local necrosis. Failure of necrosis to appear in human bites has been credited to early administration of corticosteroids, however, controlled experiments with inoculations of measured doses of venom into animals have shown no

Closure: One Stage

Lesion at Operation	Late Excision and Closure	Result and Comment
		Hospitalized for 7 d. after bite, developed typical gangrene of skin and fat, readmitted.
1½″ diameter chronic ulcer	Excision and primary suture 5 wks.	Had surgery, and healed promptly thereafter.
3 × 5″ slough of groin lesion	Excision and primary suture 3 wks.	Secondary infection, prolonged drainage
2 ulcers 2.4 and 3 cm. Excised skin and fat en bloc 4 cm, and 3 cm.	Excision and split thickness skin graft	Primary healing of graft
Undermining edge of granulating 10 × 5–8 cm ulcer Clean granulations on admission	Excision edge of skin, then split thickness graft on base	Primary healing of graft

value in any of these drugs.[11] The untreated bites of the spider on the skin of animals shows little or no necrosis in some, and extensive necrosis in others. The variable reactions observed in humans and animals are due doubtless to variations in dose of venom, depth and sites of inoculation and the severity of the secondary infection.

Surgical Considerations

Healing cannot occur until the gangrenous tissue separates. This may occur spontaneously, by non-operative treatment, or by surgical debridement. Most minor bites heal with office treatment. Early or wide excision is not advised in cosmetically or functionally important areas such as fingers or the face. Extensive lesions in other sites require surgical debridement to relieve pain, cure infection and permit earlier closure.

Early excision has seldom been performed but has many advantages (Cases 7, 8, 9). Early excision of the block of tissue containing the necrotoxin removes the gangrenous and potentially gangrenous tissue, prevents the spread of the toxin, permits early closure before gangrene and secondary infection develop. In experimental animals excision of the skin and the spreading venom six hours after inoculation prevented gangrene.[5] Excision is not advocated for bites in humans until necrosis appears inevitable, that is 2 to 5 days. In humans excision must include the superficial fascia which appears to be a natural barrier. In Case 9, the deep fascia appeared dull and was also excised. Early excision in the experimental animal fails if incomplete.[5] In humans, excision must include all necrosis and toxin, but need not include the widespread erythema. This subsides in 24 hours after excision supplemented with antibiotic drugs (Cases 8, 9).

Excision or debridement was performed in nine patients 14 to 21 days after injury when the limits of the necrosis were well outlined. Some lesions were debrided in stages rather than excised as a block. Healthy granulations appear first in the base after many weeks. Undermined edges of skin which persist and become indolent must be debrided. Debridement of the fat has been inadequate when the surgeon underestimated the extent or depth of the lesion. Although devitalized bowel or muscle

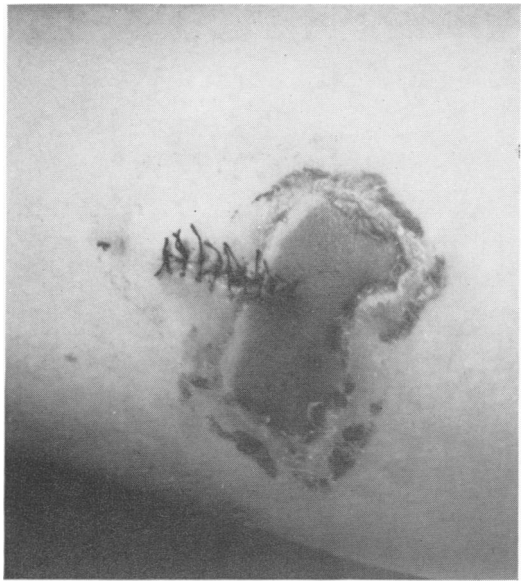

FIG. 3. Gangrenous eschar on lateral thigh 3 weeks after onset. Necrosis of the subcutaneous fat extended past the end of the biopsy incision (Case 16).

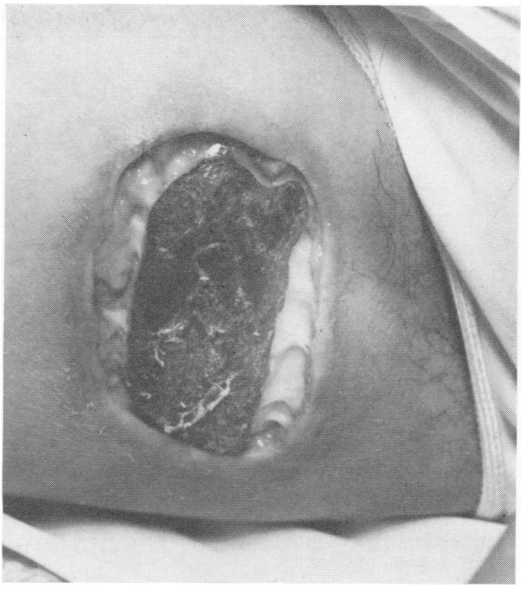

FIG. 4. Gangrenous eschar separating at edge of skin of upper medial thigh 3 weeks after onset (Case 18).

TABLE 4. *Late Debridement*

Case and Age	Spider	Site	Dates		Time	
			Bitten	Hospitalized	Operated Wks. After Bite	Healed
14. B. L. S. 37	Brown (garden)	Ankle (2 bites)	7/2	7/23 to 8/23	3½	23 d. postop.
15. M. A. G. 30	Brown *L. reclusa*	Flexor surface rt wrist	4/9	4/30 to 5/15	3	2 wks. postop.
16. B. J. M. 30	Not proven	Lateral and medial thigh	6/10	7/10 to 8/10	6	3 wks. postop.
17. L. S. 40	Brown	Buttock (inside overalls)	9/8	9/9 to 11/17	5	11 wks. scar 4–5″ diam and ½″ deep
18. H. R. 37	Brown (seen on steps where bitten)	Upper inner thigh	8/1	8/2 to 8/11 8/24 to 9/10	5	Sev. mos.

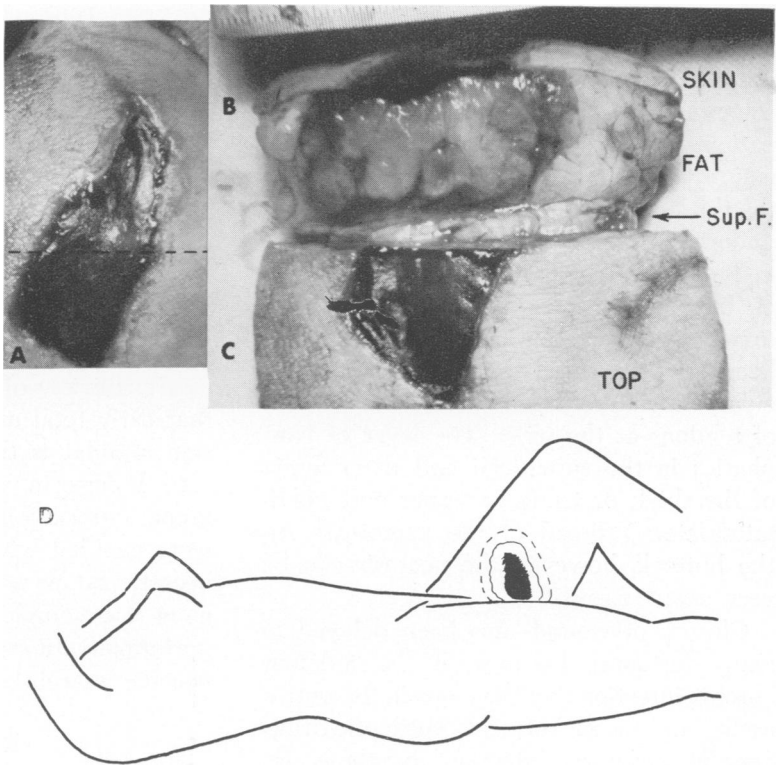

FIG. 5. Excised specimen (Case 7). Full thickness skin from upper medial thigh. A. Surface of specimen showing eschar. B. Edge of specimen cut at dotted line in A. Discolored and necrotic fat undermines skin edge 2 to 4 cm. and extends down to superficial fascia. C. Surface of specimen at line of section. D. Diagram of site of lesion. Black is the eschar. The outer dotted ring was the boundary of the excision. The inner ring was the palpable indurated zone of necrotic fat.

and Graft: Two Stages

Lesion	Operation	Result and Comment
2 ulcers, undermining edges $3 \times 3\frac{1}{2}''$ $1 \times \frac{1}{2}''$ 1 cm. deep, clean base	Debride undermined edges and connect 2 ulcers 4 d. later split thickness skin graft, $4 \times 6''$	Preliminary office care separated sloughing skin Partial loss of graft
Undermining ulcer with infected fl. carpi rad. and uln. tendons	Debride edges (general anesthesia) 3 d. later minor debridement tendons 6 d. later split thickness skin graft	Previously admitted to local hospital for 10 d Granulation tissue at base, normal skin and fat at edge
2 eschars 7×4 and 3×6 cm. undermining edge (Fig. 3)	Excise edge and down to deep fascia 3 d later split thickness graft on fat and fascia	Transferred from local hospital Partial loss of graft
Eschar 2×6 cm.	15 d. after bite, excised skin and fat $7.5 \times 5 \times 0.6$ cm. 3 d. later split thickness graft 8×15 cm. 13 d. later postage stamp graft	Complete failure of first graft due to inadequate debridement Healed by secondary intention
Ulcer 6×10 cm. (Fig. 4)	Only bedside debridement Split thickness graft	Complete failure of first graft due to inadequate debridement Healed by secondary intention

shows obvious changes in color, vascularity, etc., the devitalized fat shows only edema, poor blood supply and slight change in color (Fig. 4). Necrosis reported in human patients has spared periosteum, nerves and major blood vessels, possibly because of the thick skin and subcutaneous fat and the underlying fascia and lymphatics. In animals, however, underlying fascia and muscle have been destroyed by the necrotizing toxin.[1] Deep fascia was excised in Case 9 because it appeared thick, dull and asvascular; Case 15 required partial debridement of tendons at the wrist. The layer of lymphatics in the superficial and deep fascia of the thigh or trunk or upper arm facilitates lateral spread of the necrotoxin. In the buttock, however, the necrosis can be very deep (Case 17).

Closure of wounds has been delayed in many instances because of the indolent chronic infection that develops in the putrid eschar and the necrotic fat. Antibiotic drugs prevent invasive infection in these instances, but local infection with resistant organisms continues until all necrotic tissue is separated. We know of one instance of shock, coma, septicemia and renal failure arising in an indolent ulcer after 2 months of office treatment.[12] The skin eschar is obvious but many surgeons are not familiar with the appearance of necrotic fat. Necrotic fat, furthermore, requires weeks to slough and separate. As a result, skin grafts have frequently failed even when delayed for weeks [4] and as illustrated by Cases 15 and 18.

Primary or secondary closure by undermining the edges of skin is feasible in some patients (Cases 8, 10, 11). Around the groin, however, extensive undermining divides lymphatics and may lead to collections of lymph in the incision (Case 7). Split thickness skin grafts may be preferable at this site, and are essential for large wounds or wounds in the distal parts of extremities where the skin is not elastic or mobile. Thin split thickness skin grafts permit some contracture which diminishes the ultimate size and depth of the defect during healing.

Conclusion

Surgical considerations, pathologic features, entomology and clinical features of North American loxoscelism are reviewed.

The necrotic bite of the Missouri Brown Spider, *L. reclusa,* is sometimes so severe that hospitalization and operation are necessary. Review of 18 hospital cases reveals that early total excision of the gangrenous skin and fat is the best treatment for severe lesions in which necrosis and gangrene appear inevitable. Complications were avoided when the subtle changes in necrotic fat were recognized and debridement was complete. Grafting or closure is performed at a second stage if infection has become established.

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